54 | Headache

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Definition

Headache consists of pain or discomfort arising from pain-sensitive structures in the head. These include extracranial structures such as the skin, muscles, and blood vessels in the head and neck; mucosa of the sinuses and dental structures; and intracranial structures including the regions of the large arteries near the circle of Willis, the great intracranial venous sinuses, parts of the dura and dural arteries, and cranial nerves. The cranium, brain parenchyma, ependymal lining of the ventricles, and choroid plexus are all pain insensitive (Table 54.1).

Technique

Headache is one of the most common symptoms reported to clinicians and often causes a great amount of concern to patients. The vast majority of headaches result from benign conditions, but because the symptom can represent an early manifestation of a potentially serious disorder, it necessitates thorough evaluation. A systematic history directed to elucidate etiologic factors producing the pain is the clinician's most valuable diagnostic tool and often provides a specific diagnosis.

Headaches can be classified into three general groups based on the mechanisms by which the pain is produced (Table 54.2). Pain in *vascular headache* is produced by dilatation of cerebral arteries. *Myogenic headache,* also referred to as *tension* or muscle contraction headache, results from persistent contraction of muscles of the head and neck. *Traction headache* is caused by organic diseases involving structures in the head. The following historical topics should be addressed to classify the headache into one of these three groups.

- *Type of pain.* Many patients suffer from more than one type of headache. This may result from different etiologic factors or may represent a change in character of a chronic headache disorder.
- *Temporal profile of pain.* Acute-onset headaches of severe intensity occurring in a patient without previous history of similar headaches may suggest an organic etiology. The timing of onset and association with sleep or hormonal cycles may be helpful in diagnosis.
- *Characteristics of pain.* The location, duration, and quality of pain should be carefully evaluated. Location may be diffuse, either unilateral or bilateral, or localized to specific structures in the head and neck. Vascular headaches produce a throbbing pain; constant pain results from myogenic or traction headaches. The intensity of pain is not a reliable indicator of the seriousness of underlying conditions causing the headache.
- *Prodromes.* Neurologic symptoms may precede classic migraine headache. Visual symptoms such as scintillations, scotoma, or hemianopsia are most common; other symptoms, such as hemiplegia or ophthalmoplegia, occur rarely. Patients with common migraine may report vague premonitory symptoms such as malaise or psychic disturbances.
- *Precipitating factors.* Association of headache with environmental factors may be helpful in diagnosis. Foods such as alcohol or those containing tyramine or sodium nitrates may precipitate vascular headaches. Some patients report association with menstruation. Medications, including nitrates and other vasodilators, indomethacin, and oral contraceptives can aggravate or induce headache. Occupational factors can produce mechanical influences that aggravate headache. A history of frequent neck movements, exposure to bright lights, or long periods of work at video terminals may be helpful.
- *Associated symptoms.* Headache associated with progressive neurologic deficits or seizures can indicate an intracranial lesion. Meningeal signs occurring with an acute violent headache suggest subarachnoid hemorrhage. Migraine is commonly a "sick headache" associated with nausea, anorexia, photophobia, or sonophobia. Autonomic symptoms such as lacrima-

Table 54.1
Pain Sensitivities of Structures in the Head

	Sensitive	Insensitive
Extracranial	Skin, muscles, fascia Blood vessels Mucosa of sinuses Dental structures	Skull (except periosteum)
Intracranial	Large arteries near circle of Willis Large venous sinuses Dural arteries and parts of dura	Parenchyma of brain Pia mater, arachnoid mater, parts of dura mater Ependyma, choroid plexus

Table 54.2
Classification of Headache

Vascular headache	Myogenic headache	Traction headache
Migraine Classic Common Complicated Cluster Toxic vascular	Chronic myositis Cervical arthritis Psychogenic factors	Mass lesions Inflammatory lesions EENT diseases Arteritis Cerebrovascular disease Cranial neuritis TMJ dysfunction

tion, nasal congestion, facial flushing, or Horner's syndrome accompany cluster headaches.

- *Medical history.* Headache with onset after head trauma may suggest subdural hematoma. Previous history of malignancy or systemic disease may suggest an etiology of headache. Family history should be investigated because migraine is commonly familial. Prior investigations into a patient's headache, including attempted therapeutic interventions, should be carefully evaluated.

Basic Science

Headache can be produced by direct irritation of, or traction on, pain-sensitive intracranial or extracranial structures. Inflammatory conditions such as meningitis produce pain by direct irritation of these structures. Mass lesions including tumors and abscesses cause pain by producing traction on these structures, most commonly on dural vessels or the arteries of the circle of Willis. Factors that increase the pressure produced by such lesions will increase the intensity of pain. Headache produced by this mechanism is worsened by the increase in central venous pressure caused by the Valsalva maneuver. The patient will often note a more severe headache on awakening, which lessens somewhat after an upright position has been maintained for a period of time, due to a slight increase in brain edema induced by periods of recumbency.

The pathophysiology of migraine has been extensively studied, and the disorder is believed to result from neurogenic mechanisms. In classic migraine, evidence suggests that an initial phase of intracerebral vasoconstriction leads to focal ischemia, resulting in prodromal symptoms; this is followed by a phase of extracerebral vasodilatation, producing the headache. Changes in the metabolism of several vasoactive amines occur in association with migraine and may play a critical role in inducing the vascular changes. Plasma serotonin levels increase during the prodrome but decrease during the headache. A concomitant increase in platelet aggregation (leading to serotonin release) occurs during the prodrome, followed by a fall in platelet aggregation with the headache. Levels of other substances change during migraine and may be important in its pathogenesis (e.g., polypeptides such as bradykinin, prostaglandins, and endorphins). Local changes in vessels in the scalp occur in the headache phase and may have a role in pain production. A sterile inflammatory reaction has been observed in arteries, and the vessels display an altered reactivity to vasoactive amines.

Clinical Significance

A detailed history is of fundamental importance in the evaluation of a patient with headache and should be accompanied by a thorough physical examination. These investigations should be directed at classification of the patient's symptoms into the etiologic categories of vascular, myogenic, or traction headaches (Table 54.2).

Vascular Headache

In migraine, cluster headache, and toxic vascular headache, pain is produced by dilation of extracerebral arteries. Patients with *migraine* suffer from recurrent attacks of headaches that vary widely in intensity, frequency, and duration. The headache is commonly throbbing and unilateral in onset and may vary from side to side. It is often associated with anorexia, nausea, or vomiting. Migraine is more common in females and usually begins in childhood or adolescence. The disorder is often familial. The duration of pain is variable but usually hours to days. Some patients note an association of headache with physiologic or environmental factors.

A minority of patients with migraine experience conspicuous transient neurologic symptoms preceding or accompanying the headache. These are termed "classic" migraine headaches. The symptoms are a result of vascular ischemia in localized arterial distributions and are usually visual, but symptoms such as ophthalmoplegia or hemiplegia can occur and are termed "complicated" migraine. Rarely, cerebral ischemia in migraine can be of sufficient magnitude to produce an infarction. The incidence of stroke is increased in the migraine population. Patients with common migraine do not experience conspicuous prodromal symptoms but may report vague autonomic or psychic symptoms. Common migraine can be bilateral.

Patients with *cluster headache* experience unilateral and usually periorbital, intense and severe pain often described as burning or boring. The pain lasts from minutes to hours, often waking the patient from sleep. It occurs in clusters of weeks to months, followed by variable periods of remission. Cluster is associated with unilateral autonomic symptoms such as facial flushing, conjunctival injection and lacrimation, rhinorrhea, and less commonly, Horner's syndrome. Patients are usually males in the fourth or fifth decade, and the disorder is strongly associated with a smoking history.

Toxic vascular headache can result from many physiologic or environmental factors that produce vasodilation. Fever is the most common. Drugs including nitrates and other vasodilators, indomethacin, and oral progestational agents can cause this type of headache, as can withdrawal from pharmacologic agents such as ergots, caffeine, amphetamines, phenothiazines, or alcohol. Hypoxia, either as a result of pulmonary disease or altitude, can lead to headache.

Myogenic Headache

In *myogenic* or *muscle contraction headache,* pain is produced by contraction of muscles in the head and neck. Patients with myogenic headache have constant pain that can be located in any region in the head or neck, most often the occipital area. The pain may be reported in descriptive terms as "like a hatband" or "like a vise." Myogenic headache is extremely variable in frequency, intensity, and duration. Some patients report pain that persists for months or years, despite trials of multiple medications. Myogenic headaches often occur as a reaction to stress or as a somatic manifestation in chemically depressed patients, but can also occur in patients with cervical arthritis or following migraine attacks.

Traction Headache

A variety of organic diseases of the head can cause *traction headache.*

Headaches can result from intracranial mass lesions such as metastatic tumors, abscess, or hematoma. Pain overlies

the location of mass lesion in about half of such patients and is referred in the remainder. Masses above the tentorium frequently produce pain at the vertex or in the frontal region. Masses below the tentorium produce occipital pain; cervical muscle spasm may be present. Pain related to a cerebellopontine angle lesion is often felt behind the ear. In the presence of papilledema and raised intracranial pressure, localization of the headache is of little value. Mass lesions should always be considered in a patient with headache and papilledema or focal neurologic signs. Headache as a result of intracranial tumors often becomes more intense with Valsalva maneuvers such as coughing, urinating, or straining at stool. Such headaches are often more severe in the morning on awakening and decrease during the day as intracranial pressure decreases with maintenance of upright posture. This contrasts with myogenic headaches, which are often not present in the morning and increase in severity as the day progresses.

Conditions that cause inflammation of the meninges result in headache associated with a stiff neck and other signs of meningeal irritation. Headache occurring with a stiff neck and fever should always first suggest the possibility of bacterial meningitis. Other agents that can produce meningeal inflammation include blood, viruses, fungi, or metastatic tumor. Lumbar puncture is diagnostic and should be performed if meningeal signs are present in a patient with headache.

Diseases of ocular, aural, nasal, and sinusal or dental structures can produce headache. Headaches resulting from lesions of these structures can be identified by specific symptoms referable to those structures, or localization of pain. Evaluation of a patient with headaches should always include a thorough EENT examination.

Giant cell arteritis (temporal arteritis), polyarteritis nodosa, and less commonly arteritis resulting from other connective tissue diseases can produce headache. Headache in temporal arteritis is throbbing, intense, and persistent, and often is associated with a burning component. Patients may note pain on mastication. Ocular complaints may be the presenting symptoms. Temporal arteritis is a major and preventable cause of loss of vision in the elderly. The sedimentation rate is elevated in these conditions.

Patients with transient ischemic attacks (TIAs) or stroke can experience headache as a result of cerebral ischemia. Basilar artery insufficiency produces occipital headache, whereas carotid artery insufficiency produces pain more anteriorly.

Patients with trigeminal neuralgia (tic douloureux) experience repetitive, intense shooting pains of brief duration in the distribution of one or more branches of the trigeminal nerve. The pain is associated with trigger areas of increased sensitivity. These set off an attack when stimulated. Cranial neuralgias usually occur in middle aged or elderly patients. Rarely the glossopharyngeal nerve is involved, producing pain similar to that of trigeminal neuralgia but localized in the pharynx or tonsillar fossa and often initiated by swallowing.

Dysfunction of the temporomandibular joint can produce pain localized to the joint or referred to the jaw, neck, or along the distribution of the temporalis muscle. The pain may be associated with crepitance in the joint or limitation of jaw opening; it becomes more severe with chewing or talking.

As a general rule, acute-onset severe headaches associated with meningeal signs and headaches associated with progressive neurologic deficits are most worrisome for a neuropathologic lesion. They demand prompt evaluation. Many patients with headache will not fit clearly into any of the categories described. Reassurance, accompanied by nonnarcotic analgesics and follow-up care as needed, are often the most appropriate management.

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